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Genetics of resistance to bacterial speck of tomato caused by *Pseudomonas syringae* pv. *tomato*

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SUMMARY

Two tomato cultivars, Ontario 7710 and Rehovot 13, and their F_1, F_2, F_3 and backcross progenies were screened for resistance to bacterial speck (*Pseudomonas syringae* pv. *tomato*) of tomato. The results support the hypothesis that the resistance factors contained in the two parents are non-allelic and controlled by two different genes.

INTRODUCTION

Bacterial speck of tomato (*Pseudomonas syringae* pv. *tomato* (Okabe) Young, Dye & Wilkie) has caused increasing economic damage to tomato crops during the last decade (Goode & Sasser, 1980). In recent years some tomato cultivars and wild species which possess resistance to the disease have been found.

The resistance of the Canadian cultivar Ontario 7710 seems to be based on a single dominant gene. Two other lines from the Ontario breeding programme, 7611 and 782, possess similar resistance, whereas 20 more breeding lines showed partial resistance to the disease (Pitblado & Kerr, 1980).

Recently, two wild *Lycopersicon* accessions (*L. pimpinellifolium* P.I. 112215 and *L. hirsutum* f. *glabratum* P.I. 129157) were reported to possess a resistance factor allelic to that of Ontario 7710 (Lawson & Summers, 1982). In Turkey, two breeding lines (F_1 and F_2 Antalia) were found to be resistant, but the genetics of this resistance is unknown (Cinar, 1978). In Israel, two wild tomato lines (*L. pimpinellifolium* P.I. 126430 and P.I. 126927) were found to be resistant. The resistance of P.I. 126430 was based on a single dominant gene (Fallik *et al.*, 1983; Pilowsky & Zutra, 1982). It was also reported that 'Rehovot 13', a cultivar of Marmande type, has good field resistance (Yunis, Bashan, Okon & Henis, 1980) due to the action of a single dominant gene plus minor genes (Fallik *et al.*, 1983).

The purpose of this study was to determine whether the resistance factors of the Canadian cultivar Ontario 7710 and the Israeli cultivar Rehovot 13 are allelic or controlled by different genes.

MATERIALS AND METHODS

Plant materials. Tomato lines tested were: Ontario 7710 (P_1) obtained from the collection of the Department of Field Crops and Vegetables, Faculty of Agriculture, Rehovot (55 plants); Rehovot 13 (P_2) obtained from Hazera Seed Co., Haifa, Israel (55 plants); and the following generations derived from the cross $P_1 \times P_2$: F_1 (46 plants), F_2 (358 plants), backcross to the

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Canadian parent BC_1 ($F_1 \times P_1$) (92 plants), backcross to the Israeli parent BC_1 ($F_1 \times P_2$) (92 plants). F_3 'ex resistant-phenotype' plants were derived from seeds that were extracted separately from resistant F_2 plants (three plants with D.I. of 0·0). F_3 'ex susceptible-phenotype' plants were derived from susceptible F_2 plants (three plants with D.I. 2·33). Forty-five plants of each F_3 group were inoculated. All plants were grown in sandy loam soil in the greenhouse as previously described (Fallik *et al.*, 1983). For crossings, flowers were emasculated 2 to 3 days before and pollinated at anthesis.

Disease index (D.I.) and methods of inoculation. Plants were inoculated at the sixth true-leaf stage with a suspension of *P. syringae* pv. *tomato* and carborundum as described in a previous paper (Fallik *et al.*, 1983).

Disease severity was estimated using the disease index of Yunis *et al.* (1980): 0 = no symptoms; 1 = 1–5 specks; 2 = 6–10 specks and 3 = more than 11 specks. The number of specks on the third, fourth and fifth upper leaves were counted for each plant and the mean of the three scores was used to determine the disease index of the plant. D.I. values were used instead of % diseased plants as these indicate not only presence of disease but also disease severity.

RESULTS AND DISCUSSION

Ontario 7710 (and its sister lines 7611 and 782) and Rehovot 13 are claimed to be the only resistant cultivars that have fruits, plant characteristics and yield potential acceptable for commercial purposes (Kedar & Retig, 1976; Pitbaldo & Kerr, 1980). Thus the genetics of resistance of these cultivars to bacterial speck was investigated using these parent cultivars together with the F_1, F_2, F_3 and backcross generations of the cross between them.

The disease index (D.I.) of Ontario 7710 ranged between 0 (74·5% of plants) to 1·33 (1·8% of plants) with a mean D.I. of 0·12 (Fig. 1*a*), whereas D.I. of Rehovot 13 ranged between 0 (61% of plants) to 1·33 (3·7% of plants) with mean D.I. of 0·23 (Fig. 1*b*).

The F_1 population (Fig. 1*c*) was similar to Ontario 7710 (D.I. 0·12 and maximum disease severity reaching 0·67). The D.I. of the backcross, BC_1 ($F_1 \times P_1$) to the Canadian parent (Fig. 1*d*) was identical to that of the F_1 population, while the backcross BC_1 ($F_1 \times P_2$) to the Israeli parent (Fig. 1*e*) had a maximum disease severity of 0·67 with some increase in the mean D.I. (0·18 as compared to 0·12 of Ontario 7710). The F_2 population (Fig. 1*f*) showed that few plants (3·1% of the population) showed disease severity considered as susceptible reaction (D.I. 1·67 and above). However, 67% of the plants did not show any symptoms.

The appearance of a proportion of susceptible plants in the F_2 indicates that the two resistance factors in P_1 and P_2 are not allelic. The F_2 segregation fits the expected 15 : 1 ratio assuming two different dominant genes governing resistance ($\chi^2 = 0{\cdot}048$, D.F. = 1, $P = 0{\cdot}9$).

In order to get further evidence for this hypothesis, F_3 plants grown from susceptible F_2 as well as from resistant F_2 plants were inoculated. The mean D.I. of the F_3 plants originating from 3 resistant F_2 plants was 0·25. F_3 progenies originating from 3 susceptible F_2 plants had a D.I. of 2·1, which is approximately the mean D.I. of the susceptible F_2 plants (Figs. 1*g*, *h*).

Considering phenotypic variation one could expect some overlapping between individuals possessing maximum minor genes for resistance and those with the major resistance gene from Rehovot 13, but lacking minor genes. Some part of the phenotypic variation must also result from environmental causes. Therefore only F_3 plants derived from the extreme F_2 index classes were tested.

As each of the resistant parents (Fig. 1*a*, *b*) has a few individuals with a D.I. of up to 1·33, it is obvious that resistance gene(s) do not provide complete immunity expressed as a D.I. of zero. In a previous paper (Fallik *et al.*, 1983), it was found that disease index of the resistant parental cultivar Rehovot 13 ranged between 0 (61% of plants) to 1·33 (3·7% of plants) and the mean

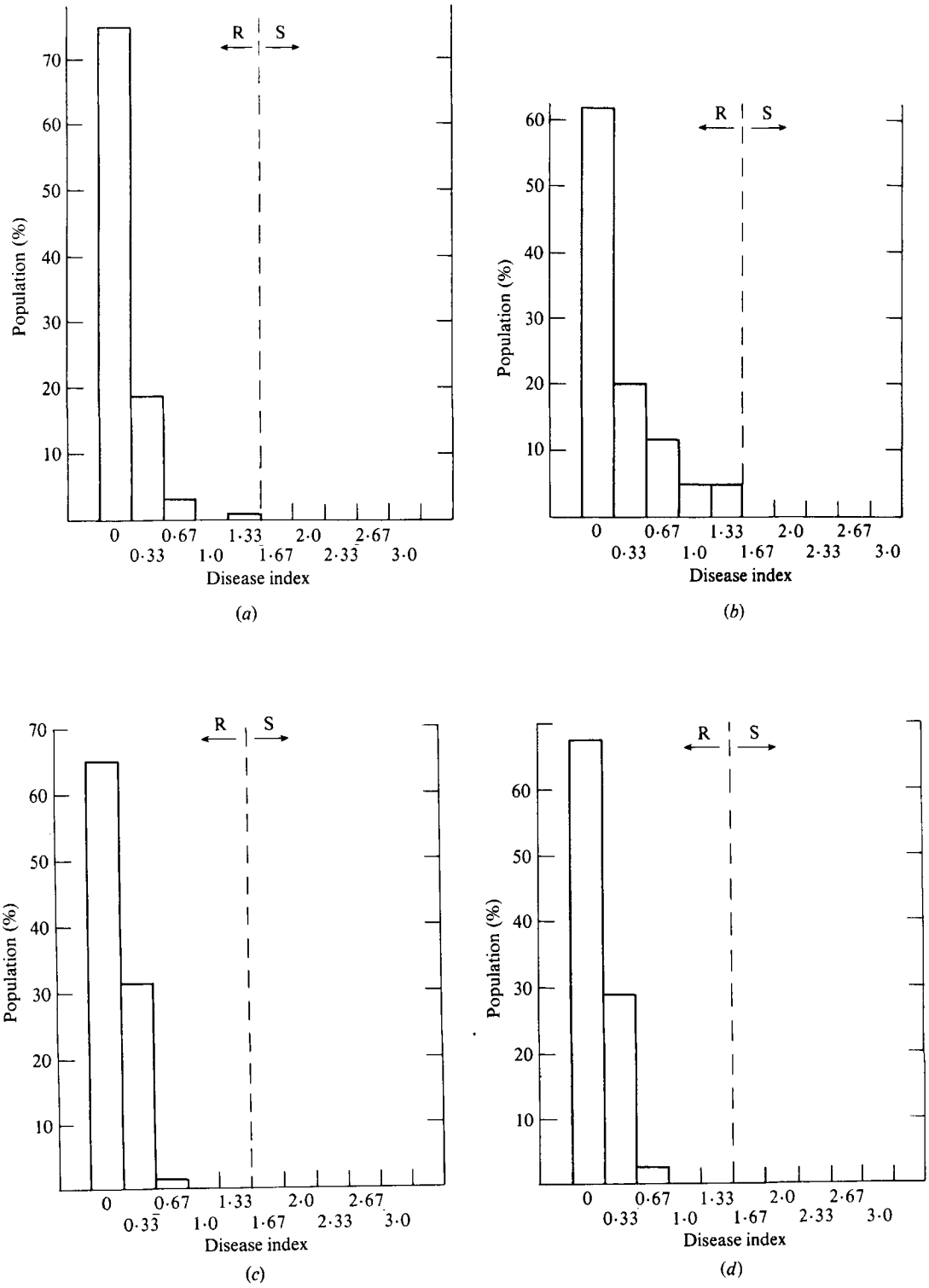

Fig. 1. Distribution of disease severity of tomato plant populations infested with *Pseudomonas syringae* pv. *tomato*. (*a*) P_1 (Ontario 7710), (*b*) P_2 (Rehovot 13), (*c*) F_1, (*d*) backcross BC_1 ($F_1 \times P_1$) to Ontario 7710, (*e*) backcross BC_1 ($F_1 \times P_2$) to Rehovot 13, (*f*) F_2, (*g*) F_3 ex susceptible-phenotype, (*h*) F_3 ex resistant-phenotype.

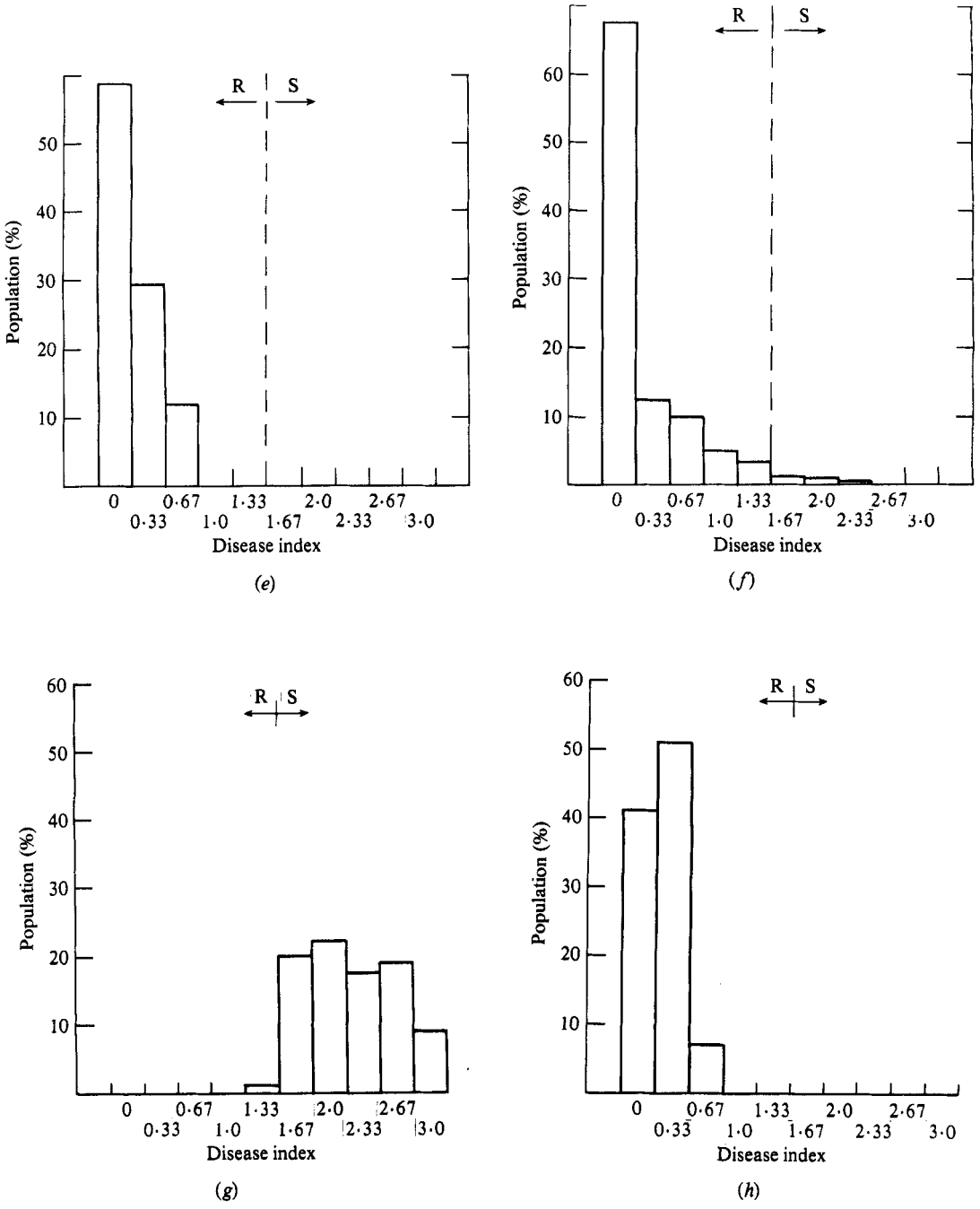

Fig. 1—*continued.*

D.I. was 0·23 whereas the susceptible cultivar VF-198 produced a disease severity ranging from 1·67 (1·7% of plants) to 3·0 (50% of plants) and the mean D.I. was 2·73. Disease indices of the parents did not overlap. Therefore, in our case, a D.I. of 1·33 indicates the limit between resistant and susceptible classes. It was shown earlier (Fallik *et al.*, 1983) that resistance of Ontario 7710 and of Rehovot 13 is governed by a single dominant gene and by a dominant gene plus minor genes, respectively. Thus, no susceptible plants should appear in the F_2 and F_3

if the genes for resistance in the two parents are allelic or identical. If, however, resistance in the two cultivars would be based on different genes, a certain proportion of the F_2 population, in our case 1/16, should be susceptible. This was apparently the case, as seen in Fig. 1(*f*) and indicated by the χ^2 of 0·048.

The mean D.I. of F_3 plants derived from resistant and from susceptible F_2 plants, 0·25 and 2·1 respectively, adds validity to the results of the F_2 population.

Resistance or D.I. in the present material is obviously a character expressed quantitatively. Nevertheless inheritance of resistance appears to result primarily from the action of major genes. Similarly, resistance to *Fusarium oxysporum* f. sp. *lycopersici* in tomatoes, a character based on a single dominant gene, is varying quantitatively as indicated by number of propagules, by percentage of infected segments or by percentage of infected plants (Alon, Katan & Kedar, 1974). Many similar examples could be cited.

The evidence found in this work shows that resistance of Ontario 7710 and Rehovot 13 is not allelic but controlled by different genes. Resistance based on the action of a single dominant gene has in many instances been overcome by appearance of new physiological races of the pathogen. If the biochemical mode of action of the two resistance genes is found to be different, the availability of different germ plasm for resistance should increase the chances of breeding tomato cultivars with long lasting resistance to the organisms causing bacterial speck.

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